

Opinion Article

Prognosis of cystic kidney disease and its types

John Francis*

Department of Nephrology, Medical University of Heidelberg, Heidelberg, Germany.

Received: 09-Nov-2022, Manuscript No. IJUN-22- 87224; Editor assigned: 11-Nov-2022, Pre QC No: IJUN-22- 87224 (PQ); Reviewed: 28-Nov-2022, QC No: IJUN-22- 87224; Revised: 05-Dec-2022, Manuscript No: IJUN-22- 87224 (R); Published: 12-Dec-2022

ABOUT THE STUDY

The term "Cystic Kidney Disease" refers to a wide variety of hereditary, developmental, and acquired disorders by the addition of neoplasms with cystic alterations. Over 40 categories and subtypes have been established. Depending on the classification, the condition may manifest as early as birth and often later in life. Cystic disease may affect one or both kidneys, and it may or may not be present in the presence of other defects. Males exhibit a higher incidence, and prevalence rises with age. More than 50% of people over 50 have been documented to have renal cysts. Cysts typically grow up to 2.88 mm every year and might result in pain and/or bleeding. The most widespread of the cystic kidney illnesses is polycystic kidney disease, which has two subtypes: Autosomal dominant, which is more common and autosomal recessive, which is less common. While Autosomal Dominant Polycystic Kidney Disease (ADPKD) is typically discovered in adults, Autosomal Recessive Polycystic Kidney Disease (ARPKD) is typically diagnosed in new-borns and young children.

Types

ADPKD and ARPKD: A variety of disorders that results in cyst formation in one or both kidneys are referred to as cystic kidney disease. The most prevalent subset is autosomal dominant polycystic kidney disease, an autosomal recessive polycystic kidney disease subset, and polycystic kidney disease, a genetic oddity with two subtypes. Therefore, the cause may be genetic, developmental, or connected to an acquired or malignant systemic condition. Simple cysts and medullary sponge kidney are two examples of acquired cystic kidney disease. Juvenile nephronophthisis, medullary cystic kidney disease, and glomerulocystic kidney disease are further forms of hereditary cystic kidney disease.

PKD: The kidneys develop many cysts as a result of PKD. These cysts contain fluid, and if they develop too rapidly, changing their shape and size, kidney injury might result. Autosomal dominant polycystic kidney disease, which is commonly diagnosed in adulthood, is caused by mutations in the *PKD1* and *PKD2* genes. These genes produce polycystic proteins, and hereditary abnormalities in these genes cause the illness known as autosomal dominant cystic kidney disease. More than 500,000 Americans have PKD, making it the fourth most common cause of kidney failure in the US. All races and genders are equally affected by polycystic kidney disease, and people who have it run the risk of having cysts in other organs such the liver, pancreas, spleen, ovaries, and big bowel. These latter cysts typically offer no issues. In the other half of individuals, symptoms may not appear at all, but they could include hematuria, back or stomach pain, or the onset of hypertension. Most cases of the disease appear before the age of 30, and 45% of patients experience renal failure by the age of 60. Autosomal recessive polycystic kidney disease, which can be detected in the womb, soon after birth, and typically before the age of 15, is considered to be caused by mutation in the *HDK1* gene.

Prognosis

The progression of polycystic kidney disease and end-stage renal disease is predicted by the number, size, and volume of cysts in the kidney. Renal cancer is not more likely to develop in people with polycystic kidney disease, but if it does, it is more likely to be bilateral. Heart problems, a burst brain aneurysm, or widespread infection are the most likely causes of mortality. Mutated gene type, gender, age of onset, high blood pressure, proteinuria, hematuria, UTI, hormones, pregnancies, and cyst size are a few variables that can alter life expectancy. The patient's life expectancy can be significantly increased if risk factors are managed and the condition is stabilised.

*Corresponding author: John Francis, Email: Johncis03@yahoo.com